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Cardiac troponins in chest pain can help in risk stratification

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with chronic diseases or disability are characterised by multiple pathology, non-specific presentations, a high incidence of secondary complications, and the need for intensive rehabilitation. They need a generalist approach to assessment and treatment and are poorly served by a superspecialist profession. Even doctors who specialise in caring for elderly people often prefer curing acute illnesses to using their skills in chronic disease and rehabilitation.⁵

To combat age discrimination health professionals and their institutions must acknowledge and document it and then act to eradicate it. These actions need to go on at all levels of the service, including hospital departments and general practices. The General Medical Council, the royal colleges, and specialist associations can all guide their members through the process and must recruit older users of the health service to help them. The charity Age Concern continues to lobby for legislation to outlaw age discrimination^{3,6} and also campaigns for a government inquiry into ageist practices in the NHS. Steps have already been taken to redress the imbalance of research in older people. The major research funding agencies now refuse to fund trials with an arbitrary age limit for recruitment. Longer term measures will begin at medical school, where modern teaching methods can be used to foster enthusiasm among medical students for older people and their problems. Partnerships with older people will enhance core teaching, as well as empower older health service users to shape the curriculum. Later on, all doctors could acquire the necessary skills by doing six months in geriatric medicine during training.⁷

Reshaping the health service around older patients need not be painful and can start now. Even small adjustments to the ward, clinic, or surgery can make a difference. For example, admission wards with access to a breadth of expertise are better for patients with multiple problems than direct admission to a specialist (say orthopaedic) ward. Individual doctors can also make a difference by seeking out and removing their own prejudices. More sweeping changes will have to follow, however, including: engaging older people in the commissioning and design of services; accepting that undergraduate and postgraduate training produces doctors whose aspirations don't match the needs

of their patients; finding and protecting money to pay for care of older people; returning to an emphasis on rehabilitation and convalescence; and changing the way we think. If the health service could be made fit for older people, it would be fit for everybody.

But there is no escaping the conclusion that a health service that will serve an ageing population well will need substantially more money than is available now. Older people probably bear the brunt of rationing within the health service. Many of those who fought in the second world war, rejoiced in the creation of the welfare state, and paid for it throughout their working lives now feel let down. Many are bitter that the government has failed to produce any response to Royal Commission on Long Term care for Elderly People that was published in March. It recommended that the personal care element of the package should be free and funded by taxation.⁸

There is still no consensus on where extra money for the health service should come from, but Professor Sir John Grimley Evans, a gerontologist from Oxford, who closed the conference, said there should be no further discussion of rationing until NHS funding is brought in line with other comparable European countries. The yearly average spend per head in the UK is 25% lower than the European average. If the government does not increase expenditure on the NHS substantially and if the health professions do not manage to counter ageism then the NHS may fail to meet the challenge presented by an ageing society.

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Cardiac troponins in chest pain

Can help in risk stratification

Despite a fall in the age adjusted prevalence of cardiovascular disease in the developed world,¹ the number of patients presenting with chest pain is rising. Greater public awareness of the importance of chest pain has lowered the threshold for seeking medical help, while improvements in our ability to manage acute coronary syndromes necessitate prompt and accurate identification of ischaemic cardiac pain. Most patients who present to accident and emergency departments will have non-cardiac pain and others, with ischaemic pain, will be at low risk of serious adverse events in the short term. In contrast,

many of those at high risk have no diagnostic clinical or electrocardiographic findings at presentation (about 50% of patients ultimately diagnosed as having an acute myocardial infarction, and 65% of those with unstable angina, present with non-diagnostic electrocardiograms).² The major challenge is therefore determining the risk of an individual patient.

There are two components to such risk. "Acute risk" is determined by the volume and severity of ischaemic myocardium (usually reflected in electrocardiographic changes) and the extent of myocardial injury (indicated by troponins and cardiac enzymes).

"Prognostic risk" is influenced by prior cardiac damage, confounding risk factors (such as age, smoking, diabetes, and hypertension), and the extent of underlying coronary artery disease (defined by stress testing, perfusion scanning, or coronary angiography). Currently, neither form of risk is systematically evaluated.

Traditionally patients with suspected myocardial ischaemia are admitted for "screening" tests to exclude myocardial infarction, largely based on serial measurements of markers for myocardial necrosis. Historically, the markers used were non-specific enzymes released from myocardial cells and other tissues such as skeletal muscle and liver. Recently, however, extremely sensitive and specific markers have become widely available—the cardiac troponins.

Cardiac troponin I and troponin T are components of the myocardial contractile apparatus. They are encoded by distinct genes, allowing the development of highly specific immunoassays.³ Unlike other cardiac markers, the troponins are undetectable in healthy subjects,³ so that even minor increases indicate myocardial damage.

Concurrent with the increasing sensitivity of tests for cardiac necrosis, it has become clear that classifying patients with acute coronary syndromes into those with unstable angina, non-Q wave infarction, and Q wave infarction is limited in accuracy and validity. A continuum of risk exists,⁴ but until recently the enzymes measured were too insensitive to reflect this. Cardiac troponins, however, provide an accurate measure of cardiac necrosis, and several large studies show that the risk of death from an acute coronary syndrome is directly related to values of troponin I or T.⁵⁻⁶ Conversely, patients with no detectable troponins have a good short term prognosis.⁷⁻⁸

The availability of such sensitive and specific markers imparts new opportunities. Instead of using blood tests merely to confirm or refute a diagnosis of acute myocardial infarction we can use cardiac troponins to triage patients with chest pain. Patients with positive values are at high risk of (re)infarction or death. They also seem to benefit most from treatments such as low molecular weight heparin and glycoprotein IIb/IIIa antagonists,⁹⁻¹⁰ though this observation from retrospective analyses needs to be confirmed prospectively. Likewise, it remains to be seen whether patients positive for cardiac troponins are those most likely to benefit from early coronary angiography and revascularisation.

Patients without ST elevation and with negative cardiac troponins six or more hours after the onset of pain have an excellent short term prognosis, leading to the suggestion that they might be discharged directly from the emergency department.⁷ Such a strategy has not, however, been tested prospectively. In the study by Hamm et al most patients were admitted to hospital and the favourable outcome among those with negative troponins may have been influenced by the treatment they received.⁷ Nevertheless, it seems that stable patients with non-diagnostic electrocardiograms and negative markers 6-8 hours after the onset of pain need not remain in coronary care units.⁸ One reasonable strategy may be to submit such patients to early predischARGE exercise testing, which provides additional prognostic information reflecting the extent and severity of underlying coronary artery disease.¹¹

Though cardiac troponins are undoubtedly useful in the risk stratification of patients with chest pain, they do have limitations. They take several hours to rise, peaking at 12-24 hours,⁴ so values on admission may be misleading. In patients initially negative for troponin a second assay should therefore be performed 6-12 hours later. In addition, values remain raised for up to 14 days,⁴ limiting their usefulness in diagnosing reinfarction. A further limitation relates to the standardisation of, particularly, troponin I assays, which are produced by several manufacturers and may give variable results, particularly at the lower end of their range.³ Clinicians should therefore familiarise themselves with the system and cut offs used locally.

Cardiac troponins provide limited diagnostic information. Though a positive result will usually confirm that chest pain is due to an acute coronary syndrome, raised values are also found in pulmonary embolism, cardiac failure, myocarditis, and renal failure.⁷ In all cases, however, this seems to reflect subclinical myocardial damage. Similarly, a negative cardiac troponin result does not rule out angina or ischaemic heart disease. Coronary artery disease is present in at least a third of patients with low risk clinical features and negative serum troponin I values throughout the first 12 hours of admission.¹² Thus, though their short term prognosis seems to be excellent, patients with suspected ischaemic heart disease despite negative troponins may require further investigation.

The assessment of patients with chest pain is a difficult skill, informed by clinical judgment. Cardiac troponins can, however, help in this process—not merely in the application of diagnostic labels but as a means to estimate risk and guide management.

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